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Brief report

An autosomal trisomic cell line in a wild common shrew (*Sorex araneus*)

JEREMY B. SEARLE

Department of Genetics, University of Aberdeen, 2 Tillydrone Avenue, Aberdeen AB9 2TN, U.K. *

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*Present address and address for all communication:
Department of Zoology, University of Oxford
South Parks Road, Oxford OX1 3PS, U.K.

An adult male common shrew with both a normal diploid and an autosomal trisomic cell line was detected during routine cytogenetic screening of animals collected from Compton, Berkshire, U.K., in August 1981. This shrew had a clone of cells with copies of the smallest metacentric, labelled *tu* in the nomenclature of HALKKA et al. (1974) (Fig. 1). Of the 50 bone marrow cells scored from conventionally-stained air-dry preparations, 7 had three copies and 43 had two copies of *tu* (Table 1).

The common shrew is well-known for Robertsonian chromosomal variation, both polytypy and polymorphism, and the mosaic shrew was collected from a site in the hybrid zone between two Robertsonian races, the Oxford and Hermitage (SEARLE 1984). Around this zone there is a dramatic Robertsonian polymorphism involving chromosome arms *j, k, l, n, o, p, q,* and *r* (SEARLE 1986). G-band preparations revealed that the shrew of interest was homozygous acrocentric for chromosomes *j, l, n, p, q,* and *r* and heterozygous for the Hermitage race arm combination *ko*. The expected chromosome number was 2n = 28, including two copies of *tu*, which was the karyotype most frequently recorded (Table 1). Those spreads with 2n = 29 are interpreted as complete trisomy for *tu*, while that with 28 chromosomes and three copies of *tu* and those with 27 chromosomes were presumably products of cell breakage during preparation.

There are two likely explanations for the mosaicism reported here. Firstly, it may be associated with neoplastic development; cf. in man, trisomic clones have been recorded in bone marrow samples from patients with acute myeloid leukaemia (e.g., FORD et al. 1975). Alternatively, the mosaicism may be the consequence of mitotic nondisjunction at any post-zygotic stage and unassociated with cancer; cf. in man, Down's syndome mosaics may have a clone of normal cells in bone marrow (Table 5.14 in HAMERTON 1971). The mosaic shrew was an old animal and thus cancer seems a likely possibility (no post mortem analysis was attempted). However, chromosomal changes associated with cancer are often more complex than trisomy for one element (FORD et al. 1975), while one would expect trisomy for a single element as a consequence of simple mitotic nondisjunction. Unfortunately, on the evidence available it is not possible to distinguish between the two explanations presented.

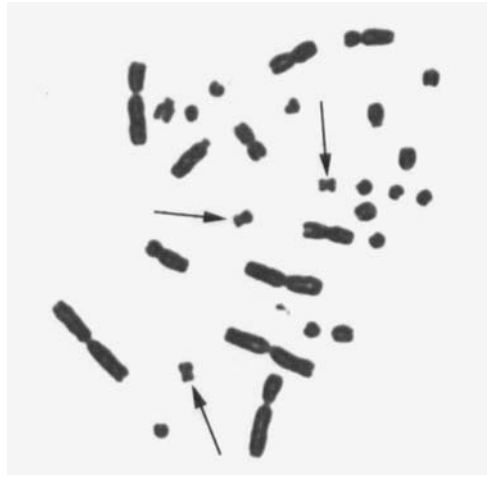

Fig. 1. A trisomic metaphase spread from the mosaic shrew. Note three copies of the small metacentric *tu* (arrows).

Table 1. The number of chromosomes and copies of *tu* in 50 conventionally stained spreads of bone marrow cells from the mosaic shrew

Number of chromosomes	27	28	29	Total
Number of spreads				
with three copies *tu*	1	1	5	7
with two copies *tu*	3	40		43

Somatic cell aneuploidy appears to be a rare phenomenon in free-living (i.e., post-partum) mammals from nature. As far as the author is aware, the only other wild-caught mammal in which an autosomal trisomic clone has been demonstrated is a water vole (*Arvicola terrestris*); FREDGA (1968) found a young female which had corneal cells trisomic for the smallest autosome. Up till the end of 1987, a total of 1337 wild common shrews from Britain had been karyotyped by the present author and coworkers, from G-banded and conventionally-stained preparations, generally based on 5–10 spreads per individual. The shrew reported here is the only example in which an autosomal trisomic cell line was demonstrated, although two cases of sex chromosome trisomy have been recorded, one a definite mosaic (SEARLE and WILKINSON 1986).

On the basis of studies in mouse and man (EPSTEIN 1986), one would expect pure autosomal aneuploidy to be highly deleterious in mammals, with all monosomies and most trisomies dying during gestation. Trisomies for certain small chromosomes in particular mammals may survive for a substantial postnatal period, and even to adulthood, e.g., trisomy for the smallest autosome in the water vole (see above: FREDGA 1968), trisomy 21 in man (Down's syndrome), trisomy 22 in the chimpanzee (MCCLURE et al. 1969), gorilla (DE GROUCHY et al. 1973), and orangutan (ANDRLE et al. 1979), trisomy 22 in ox (MAYR et al. 1985), and trisomy 28 in the horse (POWER 1987). Although not lethal, these trisomies are generally associated with phenotypic abnormalities. One would anticipate that the deleterious effect of autosomal imbalance arising from meiotic or early embryonic nondisjunction would be reduced if in addition to an aneuploid cell line there is a substantial diploid clone. Certainly in man the presence of a diploid clone ameliorates the phenotypic consequences of trisomy 21 (HAMERTON 1971). Despite this, there appear to be no previous cases of wild-caught mammals with both an autosomal trisomic and a diploid clone. This indicates either a very low frequency of mitotic nondisjunction in wild mammals and/or a high selection pressure against trisomic cells when there are also diploid cells present and/or a high selection pressure against autosomal trisomic individuals even where there is a substantial diploid cell line. It is of interest that the autosome in a trisomic condition in the mosaic shrew is one of the smallest in the karyotype and therefore is likely to have a less extreme deleterious effect in a trisomic state than trisomy for a large autosome; cf. the other non-lethal trisomies cited above. In general, one would expect individuals with neoplasms to be short-lived in nature so it is not surprising that wild mammals with aneuploid cell lines from this source have not previously been described.

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